Please cancel claims 1-19 without prejudice and add new claims 20-38 as follows:

1-19. (Canceled)

20. (New) A method for restoring cellular phenotype in a subject's cell affected by disease, damage, or age, comprising administering to the subject an effective amount of a morphogen to activate an intracellular pathway that induces intracellular formation of a Smad complex which induces expression of a phenotype-specific gene, thereby restoring the cellular phenotype in the subject's cell.

21. (New) The method of claim 20, wherein the pathway is a pathway activated by specific binding of a morphogen to its transmembrane receptor.

22. (New) The method of claim 20, wherein the Smad complex comprises Smad1 and Smad4.

23. (New) The method of claim 20, wherein the inducing step comprises phosphorylation of a Smad protein.

24. (New) The method of claim 20, further comprising inducing translocation of the Smad complex into the cell's nucleus.

25. (New) The method of claim 20, wherein the cell is a hepatocyte.

26. (New) The method of claim 20, wherein the cell is a renal cell.

27. (New) The method of claim 20, wherein the Smad complex comprises a Smad selected from the group consisting of Smad1, Smad2, Smad3, Smad4, Smad5, and Smad8.

28. (New) A method for restoring cellular phenotype in a subject's cell affected by disease, damage, or age, comprising administering to the subject an effective amount of a morphogen to inhibit TGF-β from promoting formation of scar tissue via fibrosis, thereby restoring the cellular phenotype in the subject's cell.

29. (New) The method of claim 28, wherein the morphogen activates a Smad protein.

30. (New) The method of claim 29, wherein the Smad protein is Smad6 or Smad7.

31. (New) A method for restoring cellular phenotype in a cell affected by disease, damage, or age, comprising contacting the cell with an effective amount of a morphogen to activate an intracellular pathway that induces intracellular formation of a Smad complex which induces expression of a phenotype-specific gene, thereby restoring the cellular phenotype of the cell.

32. (New) A method for restoring cellular phenotype in a cell affected by disease, damage, or age, comprising contacting the cell with an effective amount of a morphogen to inhibit TGF-β from promoting formation of scar tissue via fibrosis, thereby restoring the cellular phenotype of the cell.

33. (New) The method of claim 31 or 32, wherein the cell is a soft tissue cell.

34. (New) The method of claim 31 or 32, wherein the cell is a renal cell.

35. (New) A method for inhibiting TGF-β mediated fibrosis of tissue comprising contacting the tissue with an effective TGF-β inhibiting amount of a morphogen, thereby inhibiting fibrosis of the tissue.

36. (New) The method of claim 35, wherein the tissue is a soft tissue.

37. (New) The method of claim 35, wherein the tissue is a renal tissue.

38. (New) The method of any one of claims 20, 28, 31, 32 and 35, wherein the morphogen: (1) has at least 60% amino acid sequence identity with the C-terminal seven cysteine domain of human OP-1; (2) has at least 70% amino acid sequence homology with the C-terminal seven cysteine domain of human OP-1; or (3) is selected from the group consisting of OP-1, OP-2, OP-3, BMP-2, BMP-3, BMP-3b, BMP-4, BMP-5, BMP-9, BMP-10, BMP-11, BMP-12, BMP-13, BMP-15, DPP, Vgl, Vgr-1, GDF-1, GDF-2, GDF-3, GDF-5, GDF-6, GDF-7, GDF-8, GDF-9, GDF-10, GDF-11, GDF-12, 60A, NODAL, UNIVEN, SCREW, ADMP, and NEURAL.

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